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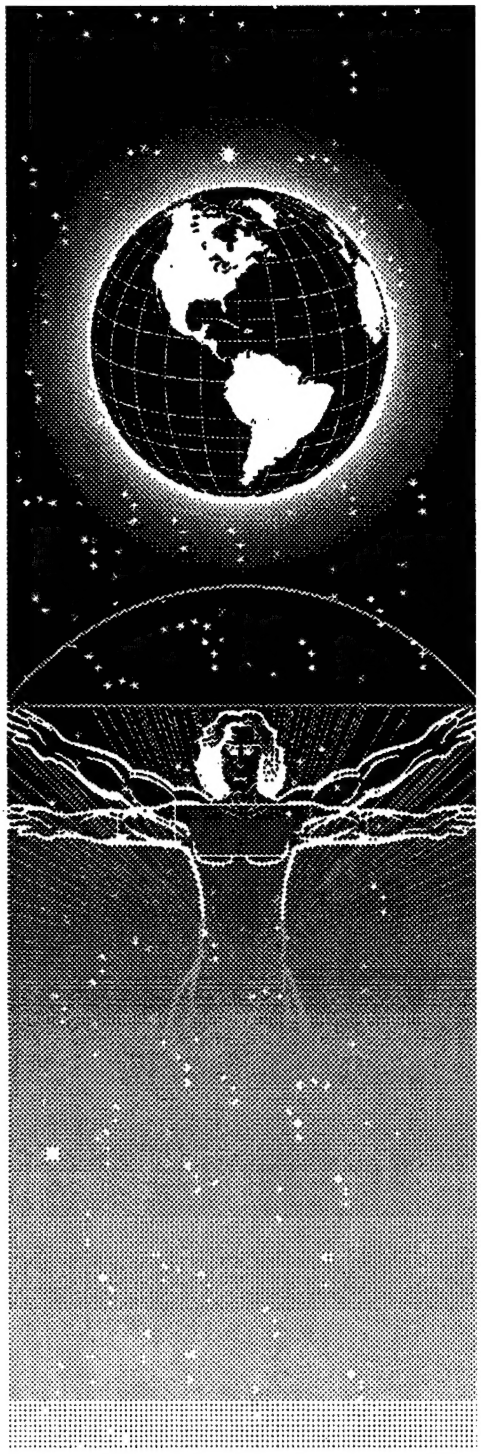

UNITED STATES AIR FORCE RESEARCH LABORATORY

THE EFFECTS OF HYPOBARIC HYPOXIA ON PSYCHOPHYSIOLOGICAL MEASURES OF COGNITIVE FUNCTIONING AND PERFORMANCE

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INTERIM REPORT FOR THE PERIOD MARCH 1994 TO MAY 1995

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The voluntary informed consent of the subjects used in this research was obtained as required by Air Force Instruction 40-402.

This technical report has been reviewed and is approved for publication.

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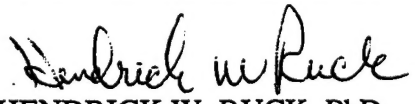

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Of special concern to the field of aviation and flight safety is the study of the effects of acute hypoxia resulting from a decrease in ambient oxygen at high altitudes. The purpose of this study was to investigate changes in brain wave activity associated with the decrements in complex task performance that are evidenced at extreme altitude when the supply of airborne oxygen is diminished. Ten Air Force personnel participated and multiple physiological measures were recorded as subjects performed a complex task designed to assess those mental functions associated with flying an aircraft. Subjects were decompressed singly via hypobaric chamber to altitudes ranging from 5,000 ft to 25,000 ft and recordings were obtained during hypobaric normoxic, hypobaric hypoxic, and recovery conditions at each altitude. Results are discussed with respect to decreased task performance and EEG metrics.

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PREFACE

This effort was conducted by the Human Interface Technology Branch (AFRL/HECP), Human Effectiveness Directorate (AFRL/HE) of the Air Force Research Laboratory, Wright-Patterson Air Force Base, Ohio. The project was completed under Work Unit 71841425, "Operator Workload Assessment." Logicon Technical Services, Inc. (LTSI),. Dayton, Ohio, provided support under contract F41624-94-D-6000, Delivery Order 0004. Mr. Donald Monk was the Contract Monitor.

The data were collected with the cooperation of the 645th Medical Group, Wright-Patterson Air Force Base, Ohio who generously permitted access to the hypobaric chamber. The authors wish to acknowledge the support of George Reis, Penny Fullenkamp, Chuck Goodyear and Barbara Palmer of LTSI during data collection and analysis.

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INTRODUCTION

In 1878, Paul Bert, the father of modern high altitude physiology, demonstrated that oxygen deficiency resulted in specific syndromes which were due to the decrease in the partial pressure of oxygen caused by a reduction of the total barometric pressure from either chamber decompression or mountain ascension. Because oxygen pressure is diminished in direct proportion to the reduction in atmospheric barometric pressure at high altitudes, the ability to transport oxygen from the atmosphere to the cell is diminished (for a review of the oxygen transport system, see Fulco & Cymerman, 1988). Since an adequate and continuous supply of oxygen is necessary to maintain cellular integrity, the resultant hypoxemia (reduced blood oxygen levels) means a reduction in the amount oxygen supplied to the brain, working muscles, and other parts of the body. Consequently, exposure to hypoxic (reduced ambient oxygen) environments has profound effects on cognitive and sensory processes, circadian rhythms, and physical endurance (Banderet and Burse, 1991). The administration of oxygen to a hypoxic subject usually results in a complete recovery as does an increase in environmental pressure that restores normal levels of utilizable ambient oxygen.

The dangers inherent in high altitude environs have been noted since the earliest experiences dealing with hypobaric chambers, high altitude balloon flights, and motorized aviation (Houston, 1987). Before the dangers were fully understood, balloon flights were sometimes fatal (Fisher, 1982) and hypoxia related errors in judgment and diminished mental capacity are also believed to have been a major cause of deaths during climbs of mount Everest (West, 1986). In high terrestrial settings like mountain climbing, some degree of acclimatization

occurs over a period of days or months that can minimize the consequences of oxygen deprivation (for a review, see Cudaback, 1984). In a flight environment, however, the amount of accommodation that can be achieved in minutes or even in hours is severely limited.

Within the field of aviation, there were few hypoxia related incidents reported until the service ceiling of motorized aircraft exceeded 20,000 ft. But, after the entry of USA into World War II, as more aircraft were designed for high altitude flight, thousands of men were exposed daily to the hazards. From the extensive collection on file at the Army Institute of Pathology, Lewis and Haymaker (1948) studied 75 cases in which death was attributed to high altitude hypoxia. To help reduce the risks, current guidelines mandate supplemental oxygen or pressurization to maintain effective cabin altitudes below 2438 m (8000 ft) to ensure crew performance. And, because this is an area of special concern to military aviation, all branches specifically address the issue in their service manuals (see Cudaback, 1984). However, these standards cannot insure that errors or accidents resulting from the sudden loss of available ambient oxygen are totally eliminated.

When challenged by an insufficient oxygen supply, a cascade of compensatory bodily responses is activated. One of the body's first defenses is the hypoxic ventilatory response (HVR), an increase in respiratory tidal volume (breath depth or amplitude) brought about by acute hypoxia. The result is a more complete turn-over of alveolar gases (CO2) with atmospheric air. Rahn and Otis (1947) reported that the HVR does not usually begin until ambient oxygen is reduced to a level equivalent to an altitude of 3000 m (9,850 ft). and studies have demonstrated that if the exposure is extended or if the level of hypoxia is more severe, then breath rate also increases (Gale, Torre-Bueno, Moon, Saltzman and Wagner, 1991; Rahn and Otis, 1947). West (1984) further reported that extreme hyperventilation is characteristic of successful mountain

climbers. Lenfant and Sullivan (1971) showed that at altitudes of 3500 m (11,550 ft), there is a net advantage to hyperventilation so that increasing hyperventilation appears to be one of the most important ways in which the body protects itself against the severe hypoxia of extreme altitudes (Lenfant, Torrance and Reynafarje, 1971).

As respiratory responses change, there is a corresponding increase in heart rate and a generalized redistribution of blood flow to protect critical body organs and functions, especially the brain. Because the brain consumes approximately 20% of the body's total oxygen intake, it is especially susceptible to injury resulting from an acute reduction in the airborne supply (Gibson, Pulsinelli, Blass and Duffy ,1981). Thus, hypoxia affects nervous tissue earlier and with greater degree of severity than it does any other tissue. In addition, there is a progression in the sensitivity of various nervous tissues to oxygen deprivation. Higher cortical centers appear to be the most sensitive followed in order by the cerebellum, medulla, spinal cord, and sympathetic ganglia. This may account for the earlier loss of higher level functioning (sensory and cognitive) relative to simple motor responses (Drinker,1938; Greene, 1957).

The specific mechanism underlying neurological deficits observed during hypoxia is unknown. Gibson. Pulsinelli, Blass and Duffy (1981) proposed that the acute effects of hypoxemia result from changes in neurotransmitter utilization and concentrations (achetylcholine, serotonin and norepinephrine) and their premise is supported by animal studies showing hypoxia related changes in hippocampal cholinergic function (Shukitt-Hale, 1993). In addition, the HVR induces vasoconstriction of the cerebral arterioles The resultant profound changes in cerebral blood flow and oxygen metabolism may account for some of the observed changes in scalp recorded brain activity. Effects evidenced by continuously recorded scalp electroencephalogram (EEG) include reduced fast wave activity coupled with increased slow

wave activity (Kraaier, Van Huffelen & Wieneke, 1988). Clinical studies have shown a progressive increase in slower brain waves, first alpha, then theta and finally delta as the severity of hypoxia increases and the EEG changes due to hypoxia are generally considered as similar to those evidenced by ischaemia (Meyer and Waltz, 1960; Gibbs, Davis and Lennox, 1935). Hockaday, Potts, & Epstein, 1965) defined 5 categories of EEGs in patients recovering from a hypoxic episode:

grade 1 : normal or almost normal record. Normal alpha with little or no theta .

grade 2 : mildly abnormal. Predominantly theta with some alpha or delta.

grade 3 : continuous delta with little faster activity.

grade 4 : periods of flattening.

grade 5 : nearly flat.

They pointed out that when delta waves become the predominant form of brain electrical activity, unconsciousness results. In a similar vein, Fowler and Kelso (1992) used visually evoked response potentials (ERPs) to assess cognitive processing during hypoxia and reported significantly increased latencies of the P300 and N200 components that were positively correlated with reaction times on a Go-No Go task. Similarly, Kida and Imai (1993) and Wesensten, Crowley, Balkin, Kamimori, Iwanyk, Pearson, Devine, Belenky and Cymerman, (1993) reported increased latencies in auditory evoked P300 components that were correlated with increased response times. Since these endogenous components are believed to reflect sensory evaluation and cognitive processing, it is clear that EEGs provide an excellent measure of hypoxia induced changes in mental functioning.

Subjectively, hypoxia has been reported to result in dizziness, headaches, visual disturbances, and on occasion, an awareness of reduced performance. Crow and Kelman (1973)

reported that even relatively small reductions in arterial oxygen saturation can significantly impair mental and motor coordination, personality, and judgment. Some individuals feel euphoric, while others become irritable and/or uncooperative. In other cases, individuals deny any detrimental effects. In susceptible individuals, measurable personality or performance decrements can occur at altitudes of as low as 3,000 m (9850 ft) (Fulco and Cymerman, 1988). However, overall impairment varies greatly between individuals probably due in part to differences in the respiratory responses. In addition to the level of hypoxia, impairment is related to factors such as: personality, motivation, experience in hypoxic environments, task requirements, task complexity, task familiarity and whether the task has been learned prior to or during the exposure (Bahrke and Shukitt-Hale, 1993; Crow and Kelman, 1973).

To determine those functions most susceptible to hypoxemia, numerous investigations have measured performance on a variety of relatively simple tasks thought to tap specific cognitive processes (for reviews see Banderet and Burse, 1991; Bahrke and Shukitt-Hale, 1993; Cudaback, 1984). For example, Kennedy et al (1989) examined changes in human cognitive and motor functions and found that hypoxia primarily affected global cognitive functioning such as intelligence and reasoning and short term memory rather than motor capacity demonstrated by simple motor tasks like finger tapping.

In an early report, McFarland (1937) suggested that visual perception and processing are particularly sensitive to the effects of hypoxia although there is some acclimatization that can occur (Kobrick, J.L., Crohn, E., Shukitt, B., Houston, C., Sutton, J. & Cymerman, 1988). Kobrick and Dusek (1970) demonstrated that a loss of visual acuity interacts with peripherally located visual stimuli to slow reaction times. In a hallmark study, Fowler and Porlier (1987) suggested that the disruption of vision influenced the response time on a serial choice task. They

further demonstrated that the response was slowed in an altitude related dose dependent manner with increasing altitudes and that there was a significant loss in function at 10,000 ft. This study provided the threshold estimate of 9750 ft for cognitive performance decrements due to hypoxia. Compared with the loss in visual acuity, auditory sensitivity appears unaffected (Heath and Harris, 1981) although significantly longer reaction times to auditory tasks during hypoxia have been reported (Kita and Imai, 1993; Wesenthal et al., 1993).

In the realm of motor functions, Shephard (1956) exposed 10 healthy individuals to a simulated altitude of 20 000 ft for 10 minutes and reported that all evidenced significant alterations in psychomotor performance, the most consistent being an increase in errors. The effects of hypoxia on mental capacity have been studied by researchers such as Denison, Ledwidth and Poulton (1966) who reported impairment of mental functions on tests for immediate memory at altitudes as low as 6000 to 8000 ft. and also on tasks performed for the first time. Others have reported similar effects at altitudes of 5000 ft (Ernsting, 1978) and 8000 ft (McFarland, 1971). Mitsubo and Akira (1993) also found that the deteriorative effects of hypobaric hypoxia on reaction time occurred abruptly at simulated high altitudes, but pointed out that individual differences in motivation improved performance and thus were a contributing factor in overall performance. This concurred with a much earlier in a report by McFarland (1937) suggesting that motivational factors contribute to performance outcomes.

Research that describes the negative effects of oxygen deprivation on complex mental activity at very high altitudes is limited, in part because of the potential risks to both participants and systems. A preponderance of studies have focused on the long term effects of insufficient oxygen on mood, behavior and cognitive skills in terrestrial settings and the degree of recovery that can be achieved through acclimatization. Others have measured performance and brain

activity at altitudes simulated by having participants inhale a gas mixture that mimics the ratio of useable oxygen at a given height. In some cases, hypobaric chambers that can simulate global atmospheric conditions have been used; but the typical behavioral tests employed have required that participants complete one relatively simple task at a time at altitudes below 20000 feet. The objective of this project was to study the effects of high altitude hypoxia on performance in relation to the EEG and peripheral psychophysiological (heart rate, eye blink, respiration, and pO2 / pcO2) measurements during different conditions (hypobaric normoxic, hypobaric hypoxic and recovery) at hypobaric simulated high altitudes (25,000 ft, 20,000 ft, 15,000 ft, 10,000 ft). The primary goals of the study were: 1) to examine the effects of acute hypoxia on complex multi-task performance, 2) to identify the concomitant physiological indicators and 3) to determine the relationship between brain activity and performance.

METHODS

Subjects and Training

Ten Air Force personnel (5 male, 5 female, ages 21-41 years) without physical disability or limitations participated in this study. All Ss had undergone recent physical examinations. All were right-handed and had normal or corrected to normal vision (20/20) vision. Two Ss were private pilots and two others had previous experience in the hypobaric chamber. Each S gave informed consent.

Before testing, Ss trained on the cognitive multi-task for two hours total in four separate sessions on two consecutive days. During training, Ss were comfortably seated in front of a computer screen, in a sound attenuated and electrically shielded chamber. Written instructions

for initializing the computer generated task were provided. They wore earphones and entered their responses using a keyboard and a joystick During the final training session, Ss wore an electrode cap and flight helmet, eye and chest electrodes, respiration bands and an oxygen mask in order to familiarize them with instrumentation procedure,

After training, Ss attended a safety seminar at the chamber facility. The multi-person capacity hypobaric chamber employed for this study can simulate a variety of global atmospheric conditions by reduction of ambient atmospheric pressure in combination with precise control of temperature and relative humidity. Low pressure atmospheres are created by removing air from the internal compartments using liquid ring vacuum pumps. The chamber was equipped with oxygen masks and helmets. Ss were briefed regarding both the procedures and the hazards of the experimental phase of the study.

Task

A PC-based flight simulation, the Multi Attribute Task Battery (MATB) developed by Comstock and Arnegard (1992) was used. The battery incorporates a set of tasks analogous to those performed by aircraft crewmembers during flight. The subtests included: systems monitoring (lights and gauges), fuel management (tanks), continuous tracking, and auditory communications. See Figure 1. Ss completed a different 3 min task scenario under each condition at each altitude. Task difficulty was not varied.

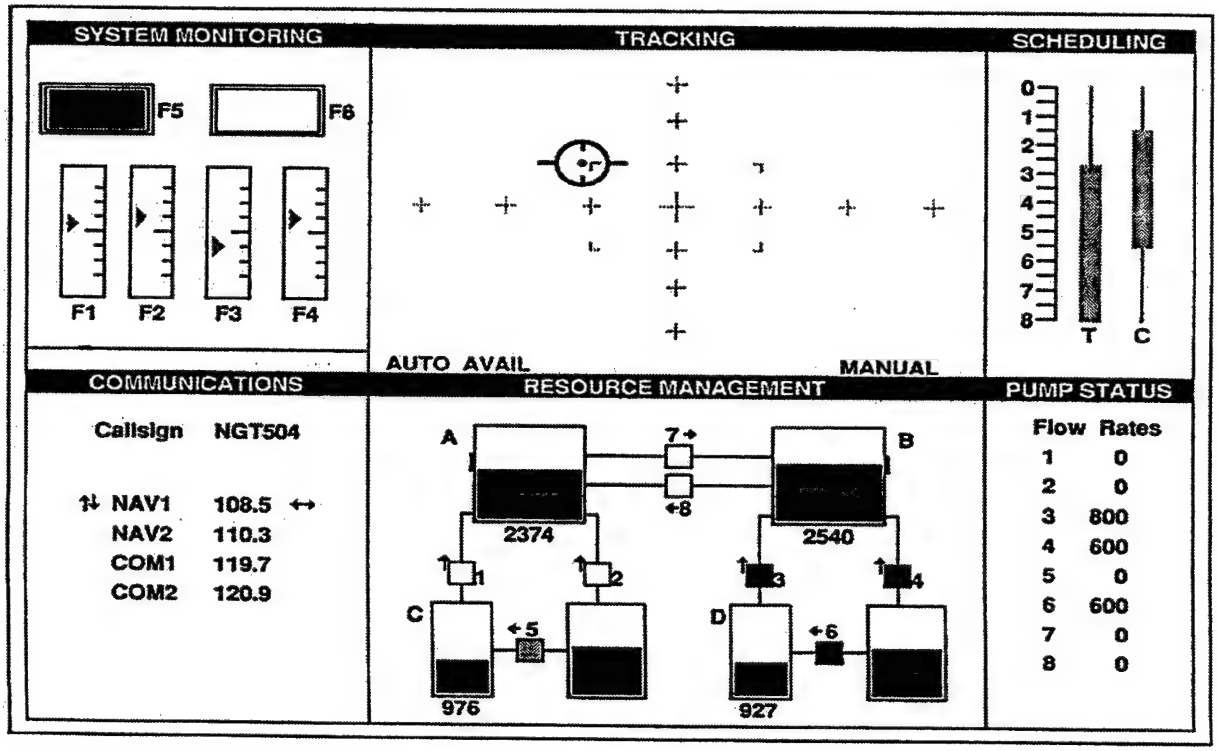

Figure 1. The MATB incorporated a set of tasks analogous to those performed by a pilot.

Systems monitoring

The monitoring task appears in the upper-left window in Figure 1. It consisted of four vertical gauges with moving pointers and green (OK) and red (WARNING) lights. The scales for the gauges were marked to indicate the temperature (TEMP1,TEMP2) and pressure (PRES1,PRES2) of the two aircraft engines. The two boxes in the upper portion of this window represented the warning lights. The light on the left was normally "on" as indicated by a green area. Ss were required to detect the absence of the green light and press the "F5" key or the presence of the red light and press the "F6" key. If the S did not detect either abnormality, the situation reverted to normal status after the preselected timeout period Normally, the green light was on and the pointers were moving within a fixed range (one line below and one line above

the center). In each 3 minute simulation, 25 malfunctions occurred when the pointer on one of the four engine gauges went "off limits " and shifted its center position away from the middle of the vertical gauge regardless of direction. Ss were responsible for detecting the pointer shifts and responding by pressing one of the corresponding function keys (T1,T2,P1,or P2) If Ss failed to detect a malfunction, the fault was automatically corrected after 10 s. Scores were based on reaction times relative to warning lights and gauges. False alarms and time outs were counted as errors.

Tracking

The demands of manual control were simulated by the tracking task located in the upper middle window. Using the joystick, the Ss task was to keep the target in the center of the window, within the dotted lines which form a rectangle. If no control input was applied, the aircraft symbol drifted away from the center toward the edges of the window. The difficulty of the tracking task (force and/or deviation) was not varied. Scores were computed based on the Root Mean Square Error (RMSE, deviation from center).

Fuel management

This task located in the lower right quadrant of the display simulated actions needed to manage the aircraft's fuel system. The display consisted of six rectangular regions. Ss were required to maintain a specific fuel level within both of the main tanks by selectively activating pumps to keep pace with the fuel consumption. Tanks were depleted of fuel at a constant rate. The deviations from the optimal fuel level over the task period were computed.

Communications

The communication task simulated receiving audio messages from an Air-Traffic Control. Ss had to recognize and respond to his/her callsign (NGT504) by executing frequency changes as instructed using the keyboard arrow keys and pressing Enter to acknowledge the change. In order to successfully complete the task, Ss had to: 1) select the proper channel (navigation vs. communication), 2) change to the requested frequency, and 3) press the Enter. Possible errors included: false alarms, incorrect frequency selection or a failure to respond before another communication was generated.

Data Collection

On the day of data collection, Ss arrived one hour prior to the experiment for instrumentation. For safety purposes, pregnancy tests were administered to all female Ss immediately before exposure to the hypobaric chamber and negative test results were confirmed. Electrodes were applied and positioned so that they did not interfere with movement or equipment and Ss were fitted with flight helmets and oxygen masks. Each S was accompanied in the chamber by a safety officer and was decompressed at a rate of 5000 ft/min and recompressed at 2500 ft/min. All Ss completed the same chamber flight profile (Figure 2) and were continuously monitored visually, acoustically and physiologically by personnel outside of the chamber.

At initial ground level (5000 ft), Ss completed a 3 min practice task. During the 30 min denitrogenation period, Ss were decompressed to 6000 ft to repudiate physiological trapped gas anomalies and verify prevention methodology. After denitrogenation, Ss performed a pre-test

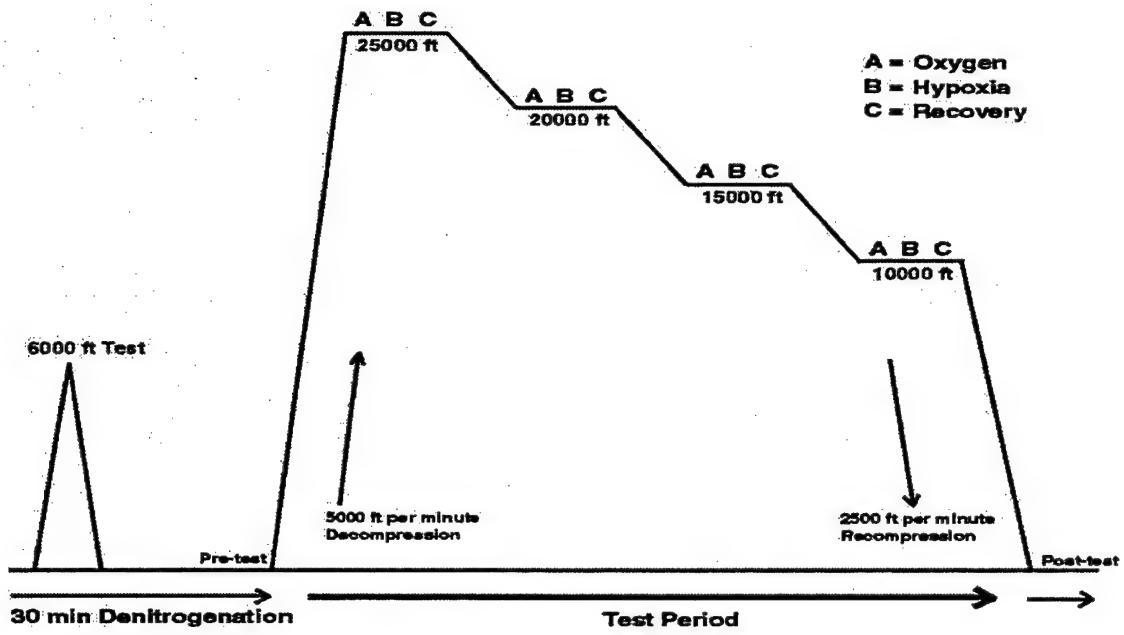

Figure 2. Subjects were decompressed singly and all completed the same flight profile.

baseline task. The chamber was then decompressed to the equivalent of 25000 ft. and after altitude was achieved, the task was performed under the 3 different conditions. They were: 1) hypobaric normoxic (100 % oxygen via mask), 2) hypobaric hypoxic (mask removed), and 3) recovery (mask replaced). After the recovery task was completed, the chamber was recompressed to the next lower altitude (20,000 ft). The same test and recompression profile was completed at each test altitude of (25,000 ft, 20,000 ft, 15,000ft, and 10,000 ft). Venting procedures were employed between the normoxic and the hypoxic conditions at each altitude to maintain normal concentrations of atmospheric gas. The total time required to complete the flight profile was approximately 1.5 hrs. Once Ss returned to ground level, a final post-test baseline task was performed. After exiting the chamber, Ss completed a physical symptoms checklist.

During testing, both vertical and horizontal electroocular activity (EOG) were monitored via Ag/AgCl electrodes (impedance < 10 KOhm) positioned above and below the left eye and at the outer canthus of both eyes. EOG signals were filtered (1-30 Hz) and amplified (x5000) using Grass P511 amplifiers. Electrocardiogram (ECG) electrodes (impedance < 30 KOhm) were positioned on the sternum and fifth intercostal space on the left side of the body. ECG signals were amplified by 2000 and filtered at 1-30 Hz (Grass P511 amplifiers). A separate ground electrode was positioned on the right side of the ribcage at the fifth intercostal space. Respiration was monitored via inductive plethysmography (Respitrace Systems: AMI Ambulatory Monitoring, Inc.). Outputs from elastic transducer bands positioned over the chest and abdomen were summed and respiration amplitudes were individually calibrated prior to data collection. EOG, ECG and respiration were sampled at 1000 Hz and stored for of-line analysis using a Psychophysiological Assessment Test System (PATS, Wilson and Oliver, 1991). Transcutaneous pCO2 and pO2 were monitored via a calibrated TCM3 microcomputer system with the sensor positioned on the right forearm. Response times for the TCM3 are 20 s for a 90% pO2 measurement and 50 s for a 90% pCO2 measurement.

EEG data were recorded from 19 active sites distributed across the scalp in accordance with the International 10-20 system (Figure 3) using a Biologic Brain Atlas III system and an Electro-Cap referenced to linked mastoids. To aid in data analysis, a synch pulse marker was stored on a separate channel. All impedances were maintained at less than 5K Ohm throughout data collection. Data were filtered at 0.1 to 30 Hz, sampled at 100 Hz, viewed on a screen during data collection and stored on disk for analysis.

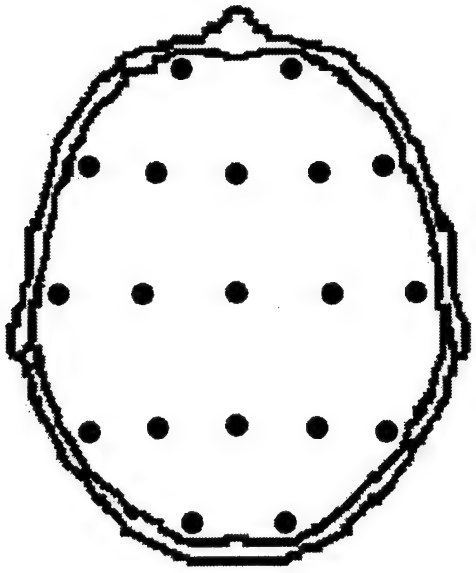

Figure 3. EEG electrodes were positioned according to the International 10-20 system.

Data Analysis

For health and safety purposes, all subjects were initially decompressed to 25,000 ft then recompressed at 5000 ft increments. Because all Ss completed the flight profile in the same order, confounding variables such as learning, fatigue, boredom, and experience could not be ruled out and altitude could not legitimately be included as a factor in the analysis. Therefore, all statistical comparisons were made between the three experimental conditions within a given altitude. Because of excessive environmental noise contamination, EEG data from one S was eliminated from the analyses. Due to technical malfunctions, EOG and ECG data were not obtained from one S and pO2/pCO2 data were not recorded from two other Ss

Performance

Subtests were scored individually based on event and response time files generated by MATB. SAS was used to calculate the tracking RMSE, fuel tank deviations, systems monitoring

(lights and gauges) reaction times and completed auditory communications reaction times. Time outs, incorrect responses, and false alarms were included in an error score. Scores were log transformed to correct for skewness before averaging across the 3 min task trial and z-scores were computed for each subtask. In addition, composite z-scores, weighted such that the continuous tracking and monitoring tasks each contributed 1/3 and the remaining discrete event scores (lights, gauges, communications, errors) each contributed 1/12 of the total, were computed. Pairwise comparisons were used to determine significant changes ($p < .05$) in performance between the hypoxic condition and the normoxic condition at each altitude.

Peripheral Physiological Measures

Digitized EOG, ECG and respiration data were reduced using PATS. Event recognition parameters (i.e., ECG R-waves and eye-blinks) were individually calibrated and visually reviewed to insure accurate identification of all events. Heart inter-beat intervals, blink rates and amplitudes, and respiration cycles and amplitudes were calculated. SAS was used to compute the average EOG, ECG, respiration and transcutaneous pCO2 and pO2 levels during contiguous 30 s intervals within a task and for the duration of each 3 min task. Statistical evaluations were performed using repeated measures ANOVAs and pairwise contrasts. All results reported as significant reached the criterion of $p < .05$.

EEG data

After application of a modified version of the eye movement correction procedure (EMCP) developed by Grafton, Coles and Donchin (1983), FFTs (resolution 0.39 Hz) were calculated over two different time periods. To assess overall changes in power, FFTs at each

electrode site were calculated based on the middle 2 min period of each 3 min task. For comparisons, FFTs were grouped into Delta (0-3.9 Hz), Theta (4.3-7.8 Hz) and Alpha (8.3-11.9 Hz) bands. Statistical analyses were conducted with SAS using separate repeated measures ANOVAs with a Geisser-Greenhouse correction for sphericity at each electrode site. Tukey's Minimum Significant Difference scores served for post-hoc evaluations of significant effects with p-values smaller than .01. Furthermore, to study changes due to hypoxia in a manner similar to that reported by Kraaier, Van Huffelen and Wieneke (1988), spectra recorded during the normoxic condition were subtracted from those recorded in the hypoxic condition and differences were statistically compared using pairwise contrasts. Significance was determined by $p < .05$.

RESULTS

Symptoms Checklist

The physical symptoms checklist that included those symptoms most commonly reported during hypoxia (i.e., headache, nervousness, euphoria etc.) was completed by each S immediately after completing the flight profile. No symptoms were reported by any subjects, under any conditions at lower altitudes. But during the hypoxia at 25,000 ft, the number of Ss reporting specific symptoms were as follows: All Ss reported feeling progressively confused and unable to concentrated, eight reported light headaches; three experienced tingling fingers; three felt nervous and one felt panic; one experienced tunnel vision and one reported that they were unable to perform.

Performance

Analysis of the z-scores obtained on the MATB subtest via pairwise contrasts with the normoxia condition indicated no significant hypoxia induced decrements in performance on the Fuel Management or Communications tasks including the number of discrete errors made even though all scores worsened under hypoxic conditions at 25,000 ft. With regard to the Systems Monitoring, there were no significant differences in reaction times to the gauges during hypoxia at any altitude but the reaction times to the lights increased significantly during hypoxia at 25,000 ($p < .05$). A significant increase in tracking RMSE also occurred at 25,000 ft. ($p < .05$). The weighted (tracking 1/3, fuel management 1/3, discrete responses and errors 1/3) composite z-score also indicated that performance deficits occurred only when Ss were deprived of oxygen at the highest altitude ($p < .05$). Below 25,000 ft, Ss were generally able to maintain their overall performance despite the lack of supplemental oxygen and corresponding signs of physiological distress. A summary of these results is presented in Figure 4.

EOG

There were no significant changes in blink amplitudes ($F = .73$ (3,24) , $p < .54$, Figure 5) or blink rates ($F = .38$ (3,24), $p < .77$, Figure 6) across test conditions at any altitude. In general, these results suggest that this response was unaffected by hypoxia and that the visual requirements of the task remained constant throughout the experiment. However, other studies have reported increases in both measures under similar conditions (Cahoon, 1970). As shown in Figure 6, during the hypoxic condition, there were non-significant decreases in blink rates at all altitudes plus increases in blink amplitudes at altitudes greater than 10,000 ft . These trends suggests that: 1) generally speaking, Ss worked harder to meet the requirements under adverse

conditions that increased visual load and 2) there was enough individual variation in their responses to render the results non-significant.

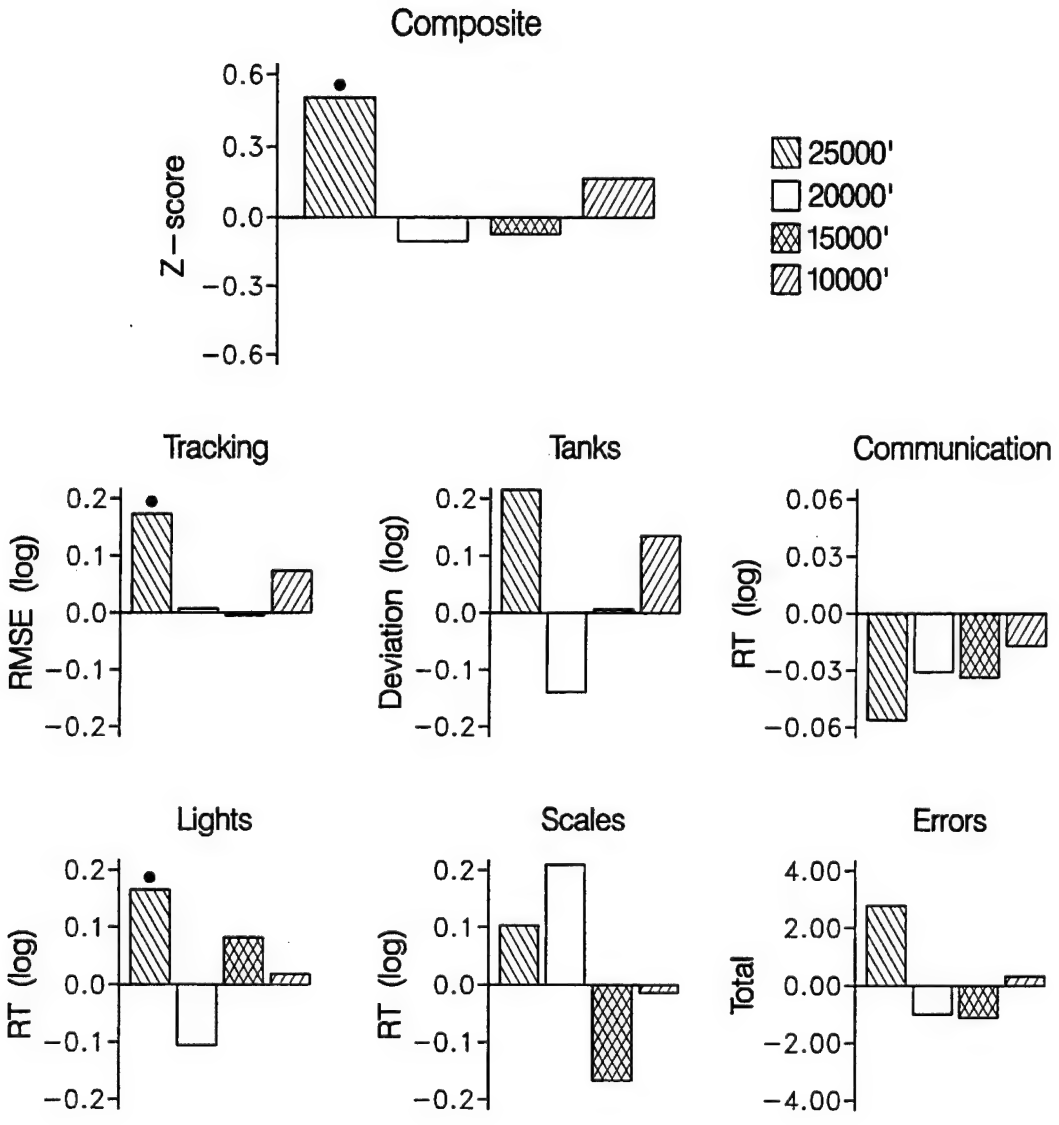

Figure 4. Significant performance decrements were only evident at 25,000 ft. Changes from prehypoxic (• = $p \leq 0.05$).

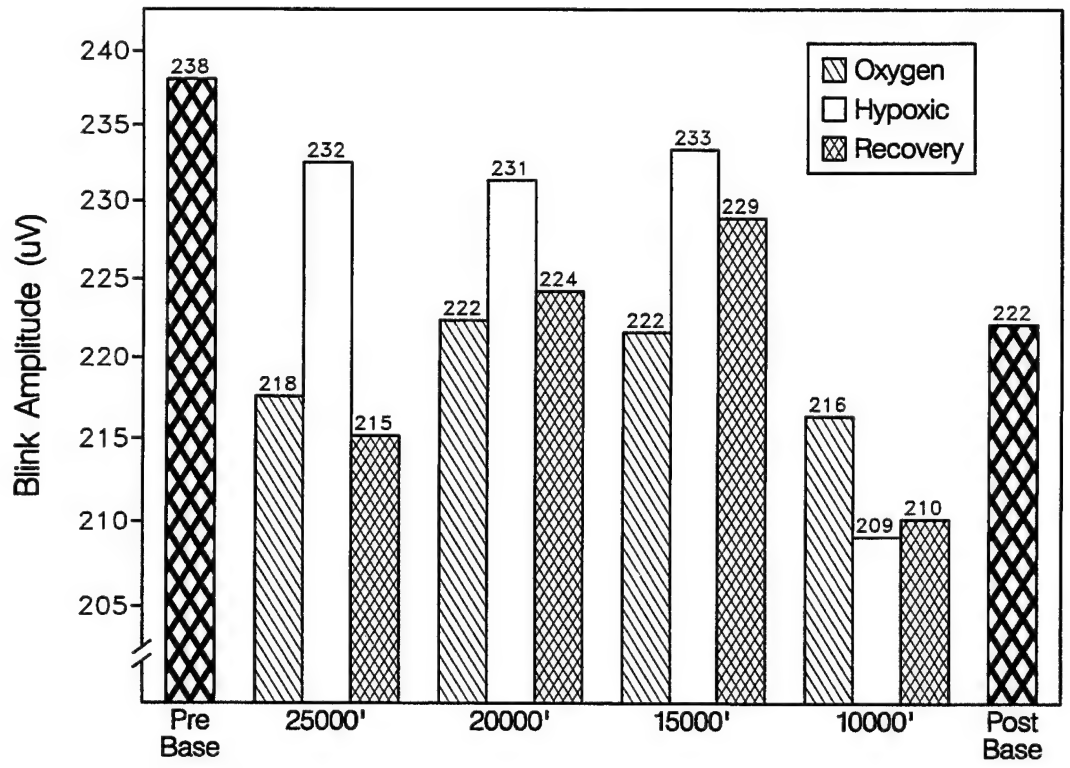

Figure 5. There were no significant differences in mean blink amplitudes between conditions at any altitude.

Respiration

Pairwise contrasts yielded significant differences in breath rates (Figure 7) and amplitudes (Figure 8) between conditions. Compared to the normoxic condition, breath rates increased during hypoxia at 10,000 ($p < .002$), 15,000 ($p < .04$), and 20,000 ft ($p < .02$) and highly increased but not significantly so at 25,000 ft ($p < .06$). During recovery, there was a corresponding significant decrease in breath rates at the same altitudes (10,000 ($p < .002$), 15,000 ($p < .04$), and 20,000 ft ($p < .03$)) but no significant decrease at 25,000 ft ($p < .06$). During hypoxia at 10,000 ft, there was also a significant decrease in breath amplitude ($p < .05$) while at

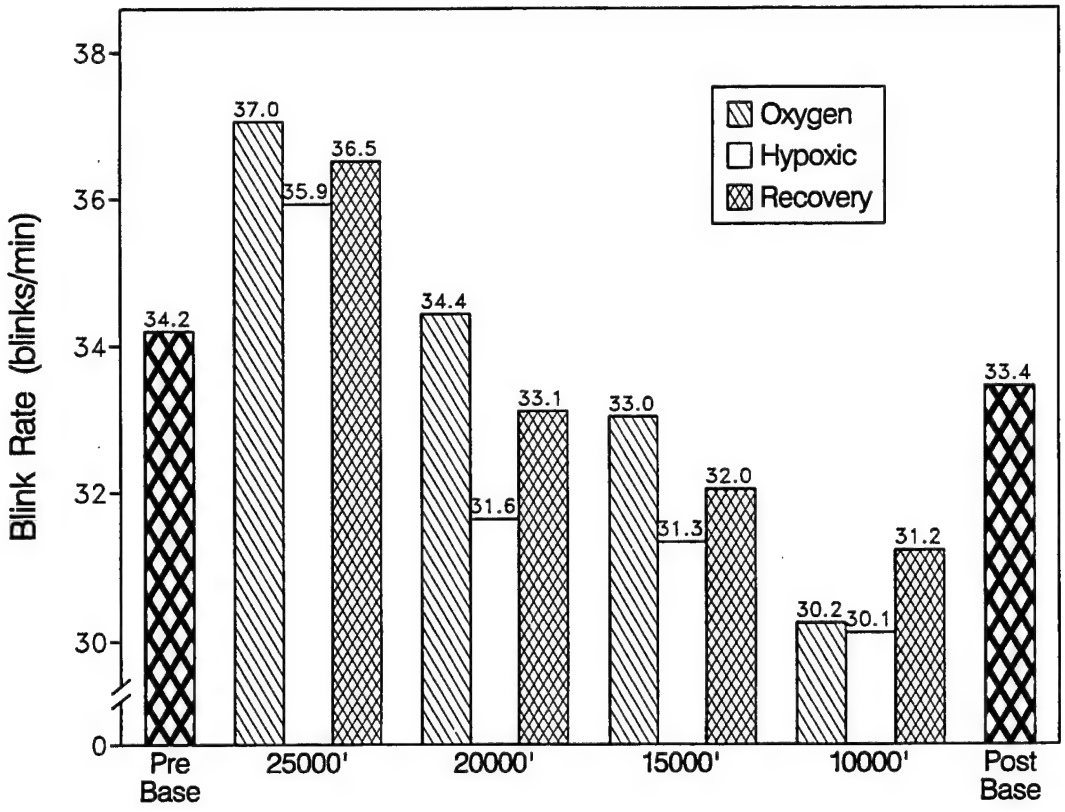

Figure 6. There were no significant differences in blink rates between conditions at any altitude.

25,000 ft breath amplitude increased significantly ($p < .01$). Examination of the data suggest that the failure to find a significant change at 25,000 ft. may have been due to the already increased breath rate during the normoxic condition at that altitude. Since it is known that stress can increase respiration rates, this increase was most likely due to the novelty of the environment and experimental conditions since this was the first test altitude for all Ss. The observed decrease in breathing rates from the pre- to post-test baseline further supports this premise even though there was no significant difference in the two baseline measures. These results support the presence of the HVR expected to be triggered by hypoxia at all altitudes greater than 5000 ft.

ECG

The average heart rate varied significantly between conditions ($F = 9.62$ (3,24), $p < .0002$, Figures 9 & 10). Heart rate increased during hypoxia at 15,000 ($p < .002$), 20,000 ($p < .0002$), and 25,000 ft ($p < .025$), but not at 10,000 ft ($p < .11$). In addition there was a significant decrease in heart rate during recovery only at 25,000 ft ($p < .025$) when the level of hypoxia experienced was more severe. Because heart rate is influenced by many factors including the concentrations of carbon dioxide and oxygen in the blood, and correspond to changes ventilation, the observed changes in HR are as would be predicted during hypoxia.

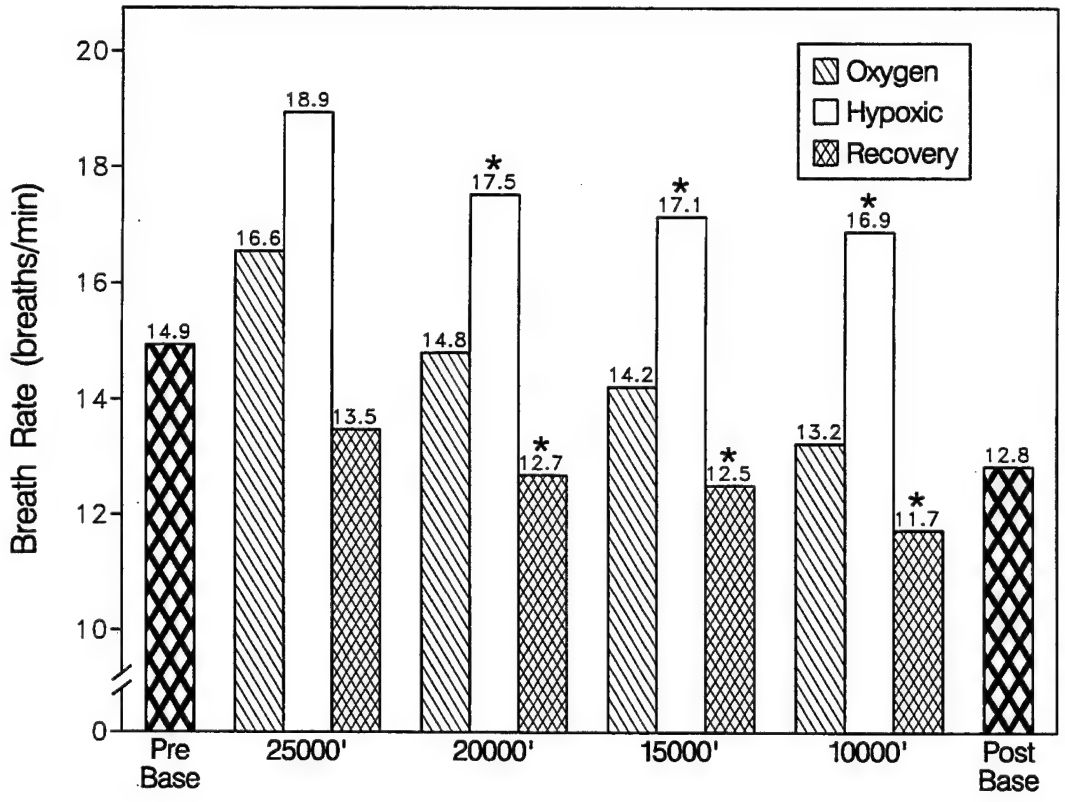

Figure 7. Breath rates increased during hypoxia and decreased during recovery. Significantly different from oxygen (* = $p \leq 0.05$).

Transcutaneous pCO2, pO2

The time course of changes in the transcutaneous pO2 and pCO2 and the statistical significance are presented in Figures 11 and 12 respectively). pCO2 differed significantly between the normoxic and hypoxic conditions at all altitudes ($p < .01$) and failed to completely return to normoxic values during the 3 min recovery period at 25,000 ft although it did return to pre-hypoxic levels during the recovery period at lower altitudes. Concomitantly, pO2 decreased

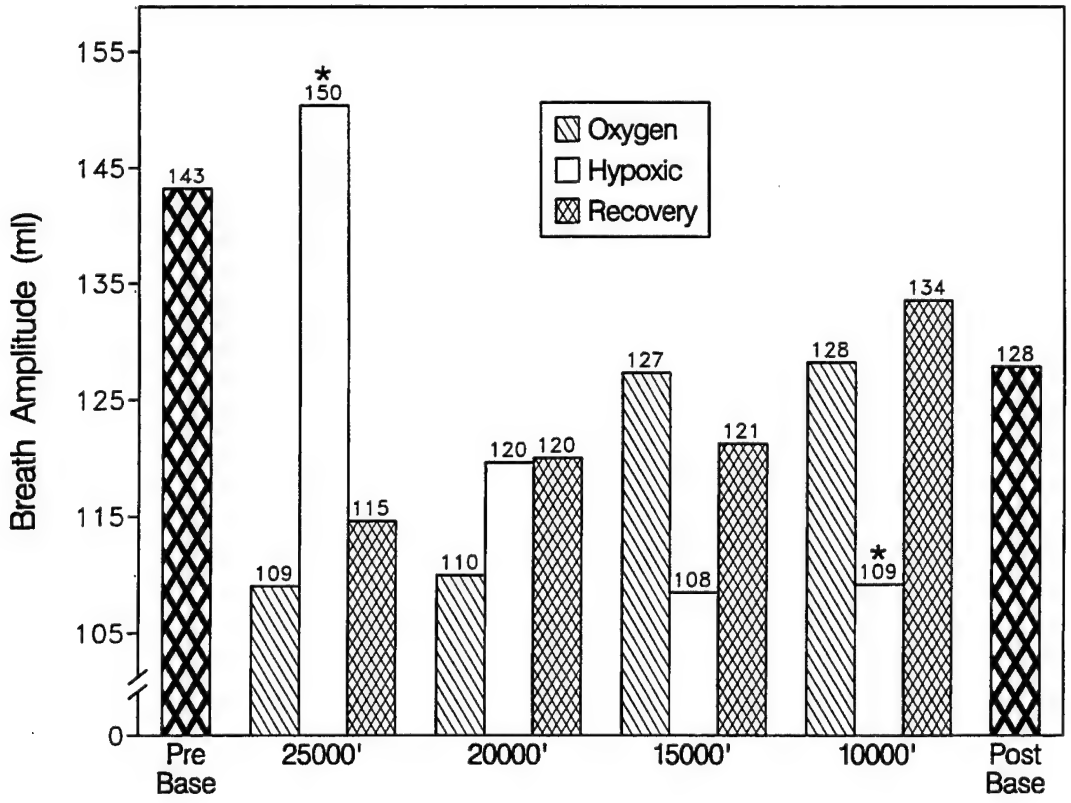

Figure 8. Breath amplitudes increased significantly at 25,000 ft and decreased at 10,000 ft. Significantly different from oxygen (* = $p \leq 0.05$).

during hypoxia compared with normoxic after 2 min at 10,000 ft, and after 1.5 min at 15,000,

20,000 and 25,000 ft. Oxygen levels then returned to the normoxic values during recovery after 1 min at 10,000, 15,000 and 20,000 ft, but it took 2 min to recover at 25,000 ft. As expected, a decrease in oxygen partial pressure, and an increase in carbon dioxide partial pressure with increasing altitude was evidenced. Furthermore, the degree of change and the prolonged recovery times demonstrated the greater physiological distress induced at the highest altitude. However, while the trends are evident, theTCM3 response time delay (20 s for pO2 and 50s for pCO2) may have contributed to what appears to be a failure of the pCO2 levels to return to normal before the recovery period ended.

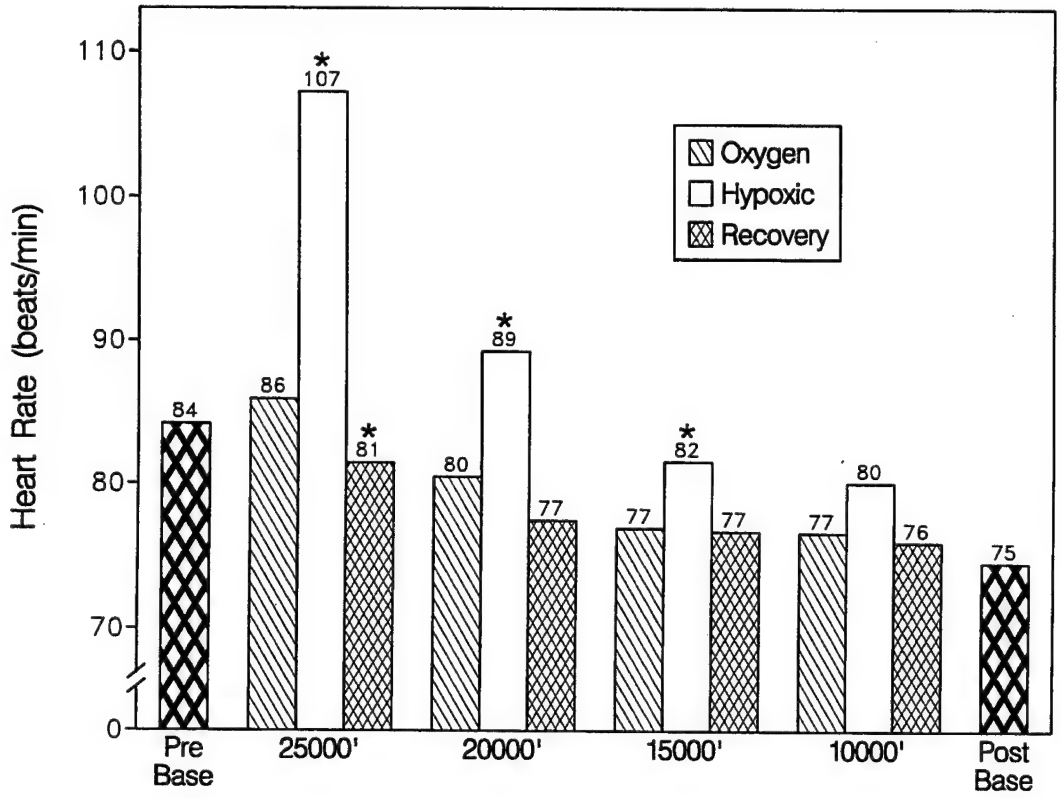

Figure 9. Average heart rates increased during hypoxia at the higher altitudes.

Significantly different from oxygen (* = $p \leq 0.05$)

10,000 ft and 15,000 ft.

At the lower altitudes, there were no significant differences in the absolute power in any of the bands between conditions nor were there any consistent differences in amount of change in

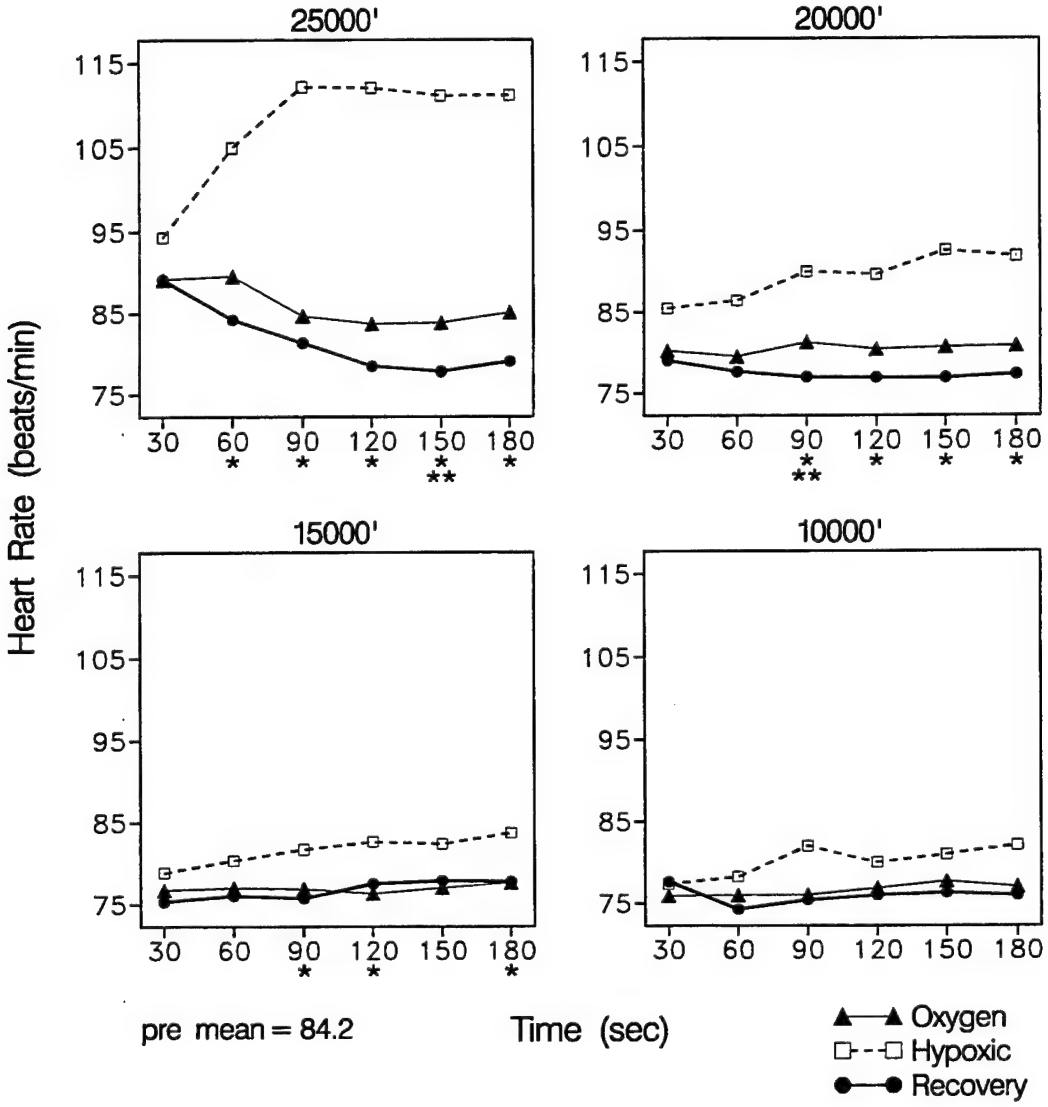

Figure 10. The time course of the changes in heart rate every 30 seconds during each 3 minute condition at each altitude. Oxy vs. Hyp, ** = Oxy vs. Rec (* = $p \leq 0.05$).

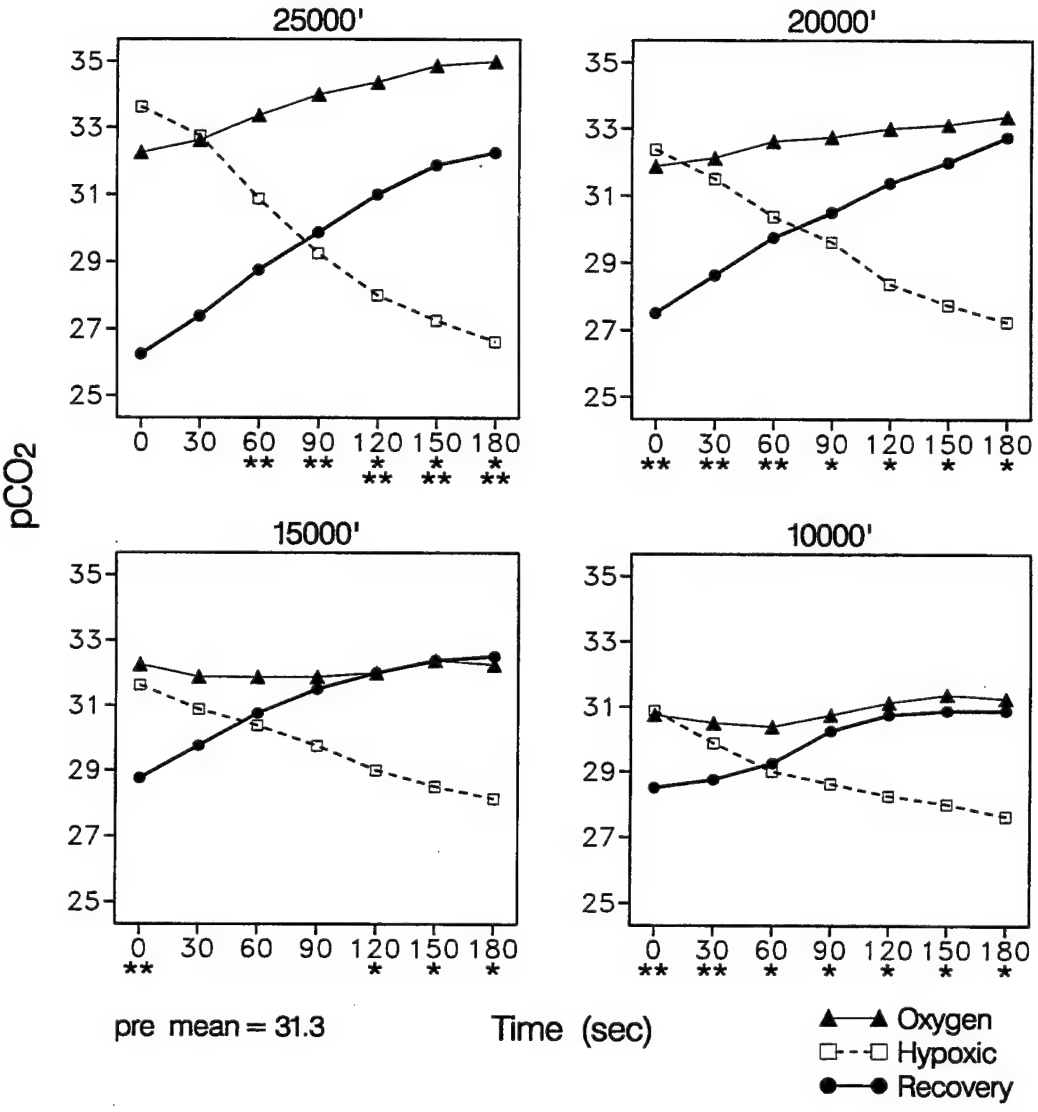

Figure 11. pCO2 levels increased during hypoxia and returned to normal within 3 min except at 25,000 ft. Oxy vs. Hyp, ** = Oxy vs. Rec (* = $p \leq 0.05$).

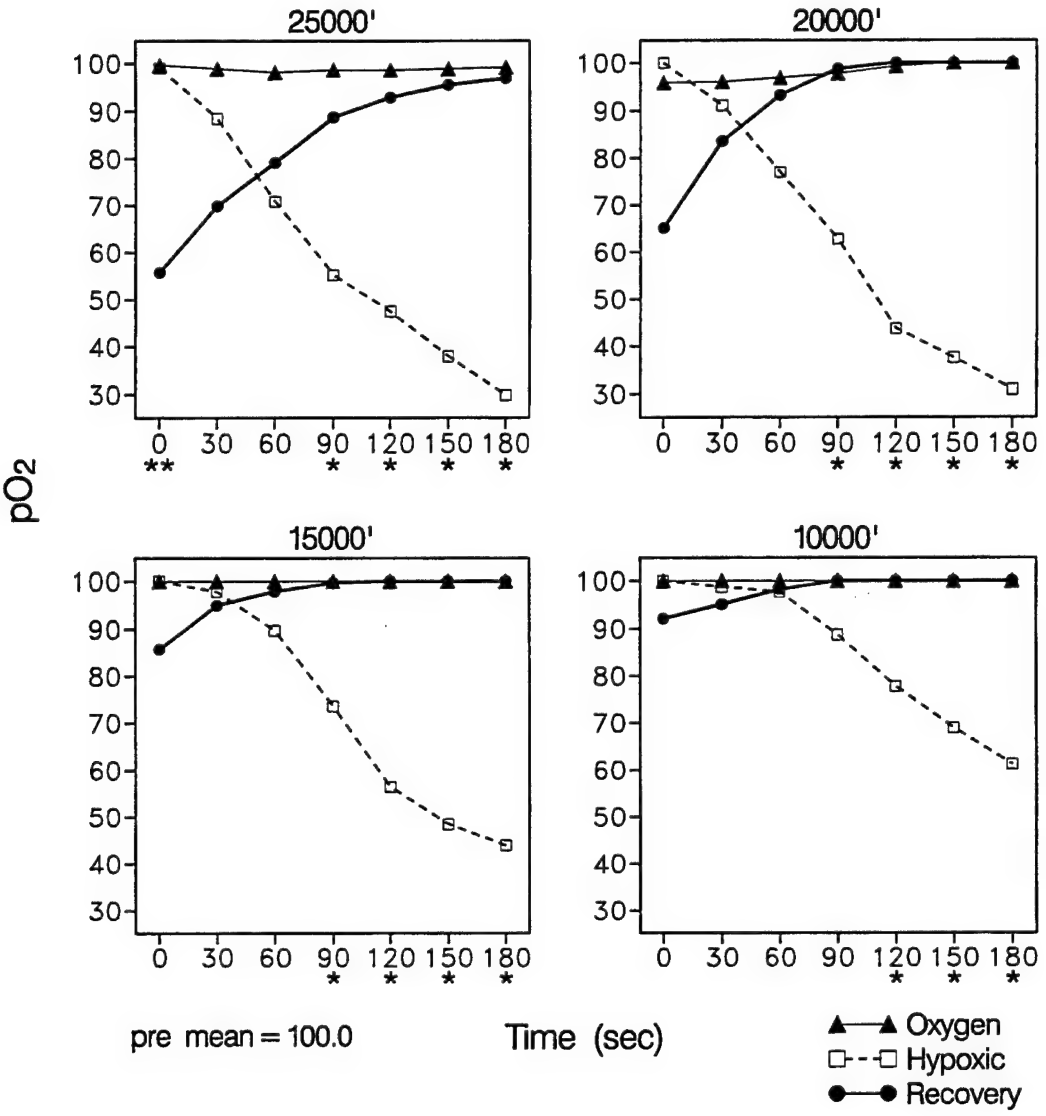

Figure 12. pO2 levels decreased during hypoxia more quickly and returned to normal more slowly at higher altitudes. Oxy vs. Hyp, ** = Oxy vs. Rec(* = $p \leq 0.05$).

any band powers that are traditionally viewed as brain wave indices of hypoxia. There were no widespread increases in either the Alpha or Theta bands and the Delta power recorded remained relatively constant at all electrode sites. However, correlational comparisons between

performance and band powers did indicated that at 10,000 ft, overall performance and performance of discrete visual monitoring tasks were positively related to the Alpha levels at numerous sites and at 15,000 ft, tracking and discrete task errors were associated with Delta, Theta and Alpha band powers at diffuse sites. It is interesting to note that measured brain wave

Table 1. At 10,000 ft, decrements in discrete visual and overall task performance were associated with changes in Alpha band power recorded at multiple frontal, central and parietal electrode sites.

Site	Delta	Theta	Alpha
Fp1			
Fp2			
F7			
F3	T		
Fz			PG
F4			PTG
F8			
T3			
C3			
Cz	G	P	PFGLC
C4			PGLC
T4			
T5			
P3			
Pz			PG
P4			PTG
T6			PTGC
O1			G
O2			PG

P = Performance Composite **T** = Tracking RMSE **F** = Fuel Management

L = Lights **G** = Gauges **C** = Communications **E** = Errors

Table 2. At 15,000 ft, decrements in tracking, the number of errors made and composite task performance were associated with changes in the Alpha, Theta and Delta band powers recorded primarily at frontal, tempero-parietal and occipital electrode sites.

Site	Delta	Theta	Alpha
Fp1		G	
Fp2			G
F7			
F3	T		
Fz			
F4	E		P T
F8			P E
T3			E
C3			
Cz			
C4			
T4		F	
T5	T	G	T
P3			
Pz		G	
P4			
T6			
O1			P T C
O2		G	

P = Performance Composite **T** = Tracking RMSE **F** = Fuel Management

L = Lights **G** = Gauges **C** = Communications **E** = Errors

($p < .05$)

activity was related to the Ss ability to maintain performance even when those values did not change significantly during the hypoxic condition compared to the normoxic conditions. For a summary of these results, see the information provided in Tables 1 and 2

20,000 ft.

There were no significant differences in the absolute power in the Delta band between conditions calculated across the 2 min period at any electrode sites. and only site T5 evidenced a significant increase in power during hypoxia compared to normoxia during the same period. There were however, significant increases in Alpha band power at several central scalp sites during the hypoxic condition relative to the normoxic condition (see Figure 13). Based on the increase in Alpha activity, at 20,000 ft, Ss evidenced changes in brain wave activity indicative of low levels of hypoxia.

Analysis of the relationship between performance and EEG measures indicated that overall performance, especially the tracking task that required continuous interaction was positively related to increases in the level of Delta, and Theta band powers at multiple sites even when the changes were not significant. Significant increases in Alpha power, on the other hand, were related to decrements in the Ss ability to maintain performance on the fuel management task (Table 3).

25,000 ft

There were no significant differences in the absolute power in the Delta band between conditions calculated across the 2 min period at any electrode sites and only site T5 evidenced a significant increase in Delta power during hypoxia compared to normoxia during the same period. There were however, significant differences in the average absolute power and in the amount of change in the Theta and Alpha band powers at numerous sites (see Figure 14). This

increase in slow activity suggests that at 25,000 ft, Ss were moderately hypoxic. In all cases, those sites that yielded a main effect for condition evidenced a significant increase in power during the hypoxic condition relative to the normoxic condition.

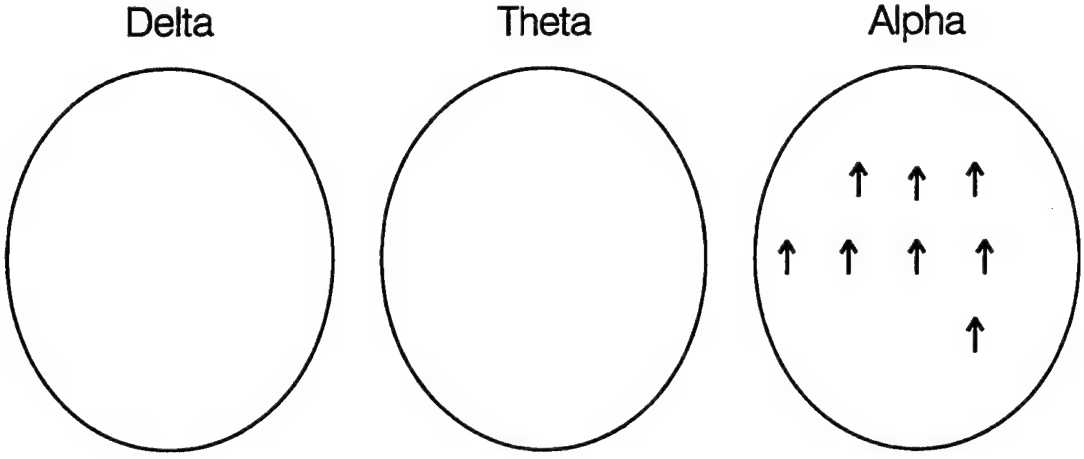

Figure 13. There were significant increases in the Alpha band power during the hypoxic condition at multiple frontal and central electrode sites at 20,000 ft. The arrows indicate increased Alpha band power at the electrode sites indicated by their position on the head ($p \leq 0.05$).

Analysis of the relationship between performance and EEG measures indicated that overall performance and especially the continuous tracking task and discrete task errors were positively related to the level of Delta and Theta band waves while increases in Alpha power were associated with poorer performance on the systems monitoring task at several electrode locations. For a summary of these results, see Table 4. It is interesting to note that the presence of Delta waves were highly related to the Ss ability to maintain performance even when the increases in Delta during the hypoxic condition relative to the normoxic conditions were non-

significant.

Table 3. At 20,000 ft, decrements in the continuous tracking, and fuel management task, plus the number of errors made, and overall task performance were associated with changes in the Alpha, Theta and Delta band powers at multiple recording sites.

Site	Delta	Theta	Alpha
Fp1	PT	PT	TF
Fp2	PE	PE	E
F7	PT	PF	TF
F3	PE	P	T
Fz		P	T
F4	PTE	P	
F8	PT	PT	T
T3		PT	
C3		P	
Cz	T		F
C4	PTE	P	FL
T4	L		
T5			
P3	T	PT	TL
Pz	TF	PTF	TFL
P4		P	FL
T6		P	L
O1			
O2		PT	F

P = Performance Composite **T** = Tracking RMSE **F** = Fuel Management

L = Lights **C** = Communications **E** = Errors ($p < .05$)

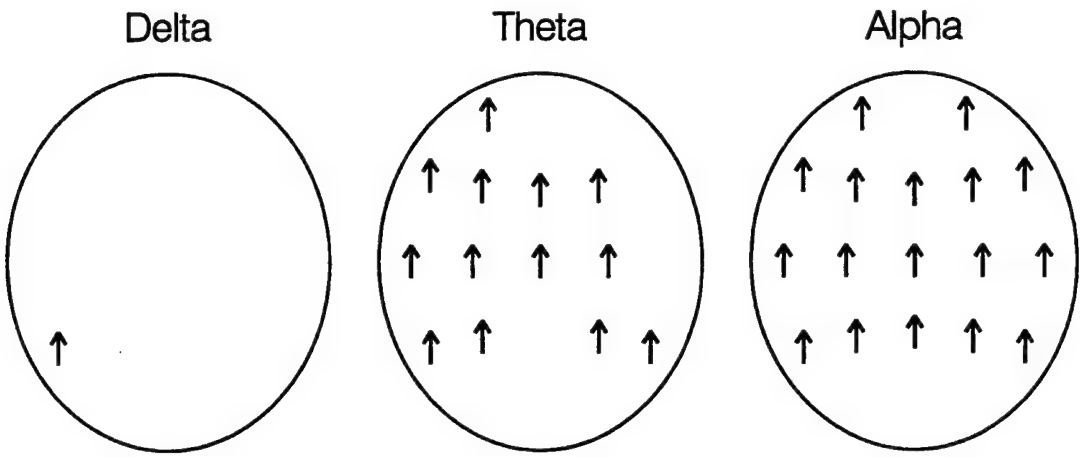

Figure 14. There were widespread increases in Theta and Alpha power during hypoxia but no absolute differences in the Delta band were evidenced. The arrows indicate increased Alpha band power at the electrode sites indicated by their position on the head ($p \leq 0.05$).

DISCUSSION

Only moderate changes in physiology and performance were observed at 10,000 or 15,000 feet. Breathing rates and heart rates increased as blood oxygen levels decreased suggesting the onset of the hyperventalitory response but no significant hypoxia related changes in brain wave activity were seen. However, there was a positive correlation between how well some discrete tasks, especially those requiring visual monitoring, were performed and the amount of change in Alpha band activity under hypoxic conditions. Evaluation of the task scores indicated that there were nonsignificant increases in the numbers of errors made and in response times, but generally speaking the participants were able to maintain their overall performance

without supplemental oxygen. Using a single-task paradigm, Fowler and Porlier (1987) estimated 10,000 ft to be the threshold level at which perceptual motor skills are grossly effected. This study, using a multiple, continuous task paradigm and highly motivated Ss failed to show any significant performance detriments at altitudes below 25,000 ft.

Table 4. At 25,000 ft, decrements in overall task performance, especially with regard to continuous tracking and the number of errors made, was associated with increased power in the Delta and Theta bands at multiple sites. Changes in Alpha were more associated with decrements in the discrete visual monitoring tasks.

Site	Delta	Theta	Alpha
Fp1	PTE	PT	E
Fp2	PTE	PE	
F7	PTE	PT	
F3	PTE	PTE	G
Fz	PTE	PTE	G
F4	PTE	PTE	
F8	PTE	PTE	
T3	PTE	PTE	
C3	PTE	PTE	L
Cz	PTE	PTE	G
C4	PTE	PTE	G
T4	PTE	PTE	
T5	PTE	PT	
P3	PTE	PT	
Pz	PTE	E	L
P4	PTE	PT	
T6	PTE	E	
O1	PTE	P	
O2	PT		L

P = Performance Composite **T** = Tracking RMSE

L = Lights **G** = Gauges **E** = Errors ($p < .05$)

At 20,000 feet, breath and heart rates rose while the blood oxygen levels dropped

significantly. FFTs of the brain activity indicated significant increases in Alpha band power at multiple frontal and central electrode sites. All physiological measures indicated that Ss were suffering the effects of hypoxia. In addition, the increased Alpha brain waves were associated with decreased systems monitoring and fuel management scores. Changes in both the Alpha and Theta powers were correlated with poorer tracking and there was also a positive relationship between changes in the power of the Delta band activity and an increase in tracking deviations and discrete task errors. The changes in FFTs were consistently associated with poorer performance even when neither measure yielded a significant change from normoxic levels. But, as was the case at lower altitudes, Ss did maintain overall performance so were able to compensate for the physiological stress induced.

The effects of hypoxia at 25000 feet, however, were much more profound. Breath rates increased and breath amplitudes rose dramatically. On the average, heart rates increased 21 beats per minute and blood oxygen levels dropped quickly to less than 1/3 of normal. Without oxygen, all participants reported feeling confused and unable to concentrate. Eight complained of headaches, three reported nervousness and tingling fingers. One reported tunnel vision and another said they felt a sense of panic and "simply gave up". There were localized increases in alpha waves that were related to tracking and monitoring errors and widespread increases in theta and delta waves that were correlated with significant increases in monitoring task errors and decreases in tracking ability.

Overall, physiological measures, including the FFTs indicated that at 25,000 ft Ss were suffering from moderate hypoxia. Correspondingly, their combined performance dropped significantly. It should be noted however that despite the peripheral physiologic indicators that all were effected, some Ss were better able to maintain their performance at pre-hypoxic levels

and compared with the other measures, the EEG data appeared to be a better indicator of performance. Evaluations of the EEG data from individual Ss indicated that in two subjects, a male pilot and a female without flying experience, the FFT spectra remained unchanged even though both evidenced severe hypoxia based on their pCO2 and pO2 levels. Alpha power increased in all other subjects. Five subjects whose performance remained relatively strong evidenced no change in Theta or Delta band activity during the hypoxic condition. Three subjects with exceptionally poor performance evidenced increases in Delta band activity while those subjects who performed well did not evidence comparable changes. The S with the poorest performance evidenced increases in all bands and further stated that they felt confused, and found it impossible to concentrate and just "gave up".

The results of this study showed that when participants were asked to simultaneously manage an array of tasks as aviators are expected to do, some types of tasks were more susceptible to the effects of hypoxia than were others. For example, under severely hypoxic conditions, participants made significantly more errors in visual-manual tracking that requires strong eye-hand coordination and in responding to lights, a skill that requires vigilance and visual acuity. This agrees with earlier reports by McFarland (1937) and Kobrick and Dusek (1970) who suggested that visual functions are most susceptible to the effects of hypoxia. In addition to the more generalized decrease, the amount of time it took to respond to red lights increased 25% during hypoxia but the time to respond to green lights increased 82%. This agrees with studies of the effects of hypoxia on night vision (Kobrick, J.L., Zwick, H., Witt, C.E. & Devine, J.A., 1984) that indicate an increased sensitivity of green receptors to decreases in ambient oxygen levels. While intriguing, this conclusion should, however, be viewed with caution since in this particular task the green light corresponded to an "off" response while the red light was associated with an

"on" response. So, the differences may also represent that difference in task requirements and not be solely dependent upon stimulus color . Of all the task components, the least effected by hypoxia was the radio communications. this auditory task was rarely missed even if the response was delayed. This concurs with other studies (Heath and Harris, 1981) that conclude that auditory functions relatively impervious to hypoxia's effects.

As the complexity of flight systems and the demands placed upon operators increases, the overall effects of high altitude flight environs must be thoroughly examined in order to properly evaluate system design and response requirements that will minimize the possibility of pilot error in the unfortunate and unexpected event of sudden oxygen loss. These data suggest that monitoring of physiological responses may hold the key. Furthermore, changes in brain activity were clearly the best indicator of even subtle changes in task performance. They more accurately reflected hypoxia's cognitive effects than did the other measures employed, including blood oxygen levels. This was especially true at the lower altitudes when overall performance did not drop precipitously. The relationship between these measures and fluctuations in mental workload are already well established and further investigations of their application in a flight environment are warranted.

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